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A CORRELATION STUDY ON THE CHRONIC
OBSTRUCTIVE PULMONARY DISEASES

BY

LYNN WALTER CAPRAUN
B.S., Florida Technological University, 1972

THESIS

Submitted in partial fulfillment of the requirements for the
degree of Master of Science: Biological Science in the
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Sciences of Florida Technological University

Orlando, Florida
1978

ABSTRACT

Data were extracted from medical records of 202 former patients of a well established central Florida general hospital. Records were selected so as to include an equal number of disease categories dispersed equally over the two years. One hundred records were dated 1973 and 102 were dated 1976. Emphysema, chronic bronchitis, and asthma had been diagnosed in 67, 67, and 68 of the cases respectively.

The age, sex, race, smoking habits, and occupations of the patients were recorded and crosstabulated with the diagnostic tests and subsequent treatment ordered by the various physicians.

Most of the emphysematous patients were males over 50, the asthmatics were females under 30, and the bronchitics were older than 50 with an even sex distribution. Most of the emphysematous and bronchitic patients had smoked over 25 years, while only 13% of the asthmatics smoked.

A majority of the chronic obstructive pulmonary disease patients complained of shortness of breath, were hospitalized ten days or less, treated four times a day with intermittent positive pressure breathing had little or no pulmonary rehabilitation, and survived.

Respiratory care appeared to improve over the three-year period as judged by an increase in the frequency of blood gas monitoring and a reduction in the required ventilator times with improved techniques.

ACKNOWLEDGMENT

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LIST OF TABLES

	Page
1. Frequency distributions of patients for year, diagnosis, sex and race.....	22
2. Frequency distributions of patients for hospital stay, patient complaints and smoking habits.....	23
3. Frequency distributions of patients for mechanical ventilation, IPPB, and type of airway.....	25
4. Crosstabulations of diagnosis by sex and by age.....	27
5. Crosstabulations of diagnosis by length of hospital stay and by occupation.....	29
6. Crosstabulations of smoker by occupation.....	31
7. Crosstabulations of diagnosis by smoking habits and by length of time smoking.....	32
8. Crosstabulations of diagnosis by mechanical ventilation and by pulmonary rehabilitation.....	34
9. Crosstabulations of diagnosis by race and by bronchospasms.	36
10. Crosstabulations of diagnosis by death.....	37
11. Crosstabulations of year by time on ventilator and by ventilator modifications.....	39
12. Crosstabulations of year by blood gases and by controlled oxygen administration.....	40

LIST OF FIGURES

	Page
1. Data sheet for medical record auditing.....	19

INTRODUCTION

Many people suffer from a group of chronic, frequently progressive respiratory disorders which appear related but have no single cause. Those patients with chronic bronchitis, asthma or emphysema who exhibit persistent obstruction of bronchial air flow are classified as having a chronic obstructive pulmonary disease (COPD).

The many different interpretations and usages of COPD have led to confusion in the medical literature and to misinterpretations in the analysis of morbidity and mortality data. Although pulmonary emphysema is a continually used clinical term, it is not always present as an anatomical circumstance when diagnosed radiologically or clinically.(1) Throughout the spectrum of chronic obstructive pulmonary disease, the complications may range from pure obstructive disease of the airway with bronchitis and no emphysema, through various combinations to severe pulmonary emphysema without significant bronchitis. The mechanisms and processes which bring about these conditions are neither static nor necessarily progressive. Therefore, all stages of disease are possible, from reversible abnormalities to relentlessly progressive cardiopulmonary insufficiency.(2,3)

In order to establish a clearer understanding of chronic obstructive pulmonary disease, one should review the normal structure and function of the lung. It should be recalled that the airways are formed early in fetal life as a centrifugal outgrowth from the floor

of the pharynx, and this growth is genetically determined. On the other hand, alveolar development occurs during the seventh intra-uterine month and occurs initially at the periphery of the respiratory tree. At birth, there are approximately 20 million alveoli, and the development of alveoli continues until the individual is approximately eight years of age and at this point the lung contains about 300 million alveoli. The alveoli will continue to grow until they mature, but they will not increase in number. This type of developmental process allows for dysanaptic growth, that is, disproportionate growth withi an organ. The situation is set for fetal injury to alter growth and development, possibly resulting in a decreased flow rate and a predisposition for COPD.(4,5)

The major supporting structure of the lung consists of elastic and collagen fibers that have somewhat of a helical shape with multiple crosslinks.(8) This configuration allows for normal expansion and recoil. This can be compared to the mechanics of a gate spring which elongates, but the length of the wire making up the spring remains the same, and the torsional strain throughout the helical arrangement of the spring closes the gate. The helical arrangement of the fibers in the alveolar ducts provide reactive forces, providing that the collagen and elastin are normal.

The alveoli represent curved surfaces with an air-liquid interface, and as a result they are subject to the effects of surface tension according to LaPlace's relationship of a sphere. Under these conditions, the pressure that is required to inflate an elastic sphere is related directly to twice the surface tension of the sphere, and

inversely to the radius. It is readily apparent that if two spheres of unequal size are connected together, that inflation will preferentially occur in the larger sphere with resultant collapse of the other sphere. If this were the situation in the lung, there would be large-scale atelectasis or alveolar collapse. This usually happens as a result of chronic obstructive pulmonary disease where obstruction and increased airway resistance causes some alveoli to break down and hyperinflate and others to collapse.

In the lung of an adult there are definite limits to the rate at which an individual can blow air out. Once maximum flow is attained, then additional patient effort does not increase expiratory flow rate. The factor that influences an individual's ability to generate gas flow is primarily airway caliber. It is one of the simplest and most obvious factors and is often overlooked. Airway caliber is dependent upon anatomic and physiologic influences such as (a) intrinsic size of the airway, which is determined genetically, and (b) the state of distention of the bronchial walls.(6) Resistance to air flow in the airways can be increased very dramatically with physiologically induced changes in the supporting smooth musculature of the airways and circulatory changes in the supporting smooth musculature of the airways and circulatory changes in the mucosal membranes that line the inside of these airways.(7)

Emphysema

Emphysema is defined as the permanent, abnormal enlargement of any part of the lung parenchyma distal to the terminal bronchiole,

accompanied by destructive changes.(2) The word emphysema comes from a Greek term which means "blow into" or "inflated." When applied to the lungs it indicates an abnormal degree of inflation and overdistention of the acinus with air that cannot be expelled due to some expiratory problem. The acinus is considered the unit structure of the lung and includes the respiratory bronchioles, alveolar ducts and the alveolar sacs or alveoli. All components of the acinus take part in gas exchange.

Though pulmonary emphysema is defined in terms describing inflation, the cause of this is fragmentation of the alveoli, resulting in a loss of lung elasticity.(3) Therefore, instead of the lungs simulating rubber balloons, they become more like paper sacs. Parenchymal tissue along with alveolar tissue are major factors in maintaining the patency of the smaller airways; therefore, when the alveoli breakdown, the walls of these small airways actually start to collapse. As the air pressure inside the lungs builds up during forced expiration, the collapse becomes worse. Patients that develop this problem tend to retain secretions in the collapsed airways. These secretions become easily infected and cause bronchial irritation, edema, bronchospasm and reduced ventilation, leading to many clinical features that are many times overlooked or misdiagnosed and as a result improper or insufficient treatment and follow-up is instituted.(9,10)

There are many types of emphysema differentiated mainly on the anatomic description. Chronic generalized obstructive pulmonary emphysema is permanent pulmonary hyperinflation with an increase in total lung capacity resulting from an increase in residual volume

and associated with smaller bronchiole obstruction throughout all pulmonary segments, with loss of lung elasticity.(7) Nonobstructive pulmonary overinflation (compensatory emphysema) results from excessive lung expansion required to occupy vacancies caused by removal of lung segments or segmental collapse. Obstructive pulmonary overinflation (localized obstructive emphysema) is a condition resulting from a "check valve" mechanism due to partial obstruction of a bronchus by a foreign body or growth, therefore making it more difficult for air to leave back out through that bronchus during expiration. Centrilobular emphysema is caused by distended and distorted respiratory bronchioles which upon examination appear as enlarged centrilobular air spaces. This type may not be detected clinically or radiologically until it is in its advanced stages. Skeletal or false emphysema is a condition found in older individuals where the ribs are fixed in an elevated inspiratory position, the thoracic spine becomes kyphotic, and the thoracic cage becomes rounded. Upon observation this condition resembles pulmonary emphysema but even though the skeletal structure is abnormal the lungs are expanding and contracting normally because of adequate excursion of the diaphragm. Bullous emphysema is caused by single or multiple large cystic alveolar dialation which may or may not be associated with chronic generalized obstructive emphysema. Subcutaneous emphysema results from air leaking into the suprasternal notch and extending into the subcutaneous tissues of the neck, face, arms and trunk.

The clinical manifestations and symptoms of pulmonary emphysema depend on how advanced the emphysema is, the type of emphysema and

the extent to which the circulatory system is affected. Alveolar destruction will increase the pulmonary vascular resistance, causing the right side of the heart to work harder. This causes dyspnea or shortness of breath which is also a characteristic symptom of pulmonary emphysema; however, it may be difficult to distinguish between dyspnea due to emphysema and that caused by heart failure induced by pulmonary resistance.(2,11) Knowledge of the patient's history may help the examining physician distinguish the difference but sometimes even the best physicians have difficulty. Dyspnea is generally noted upon exertion, but in time breathing becomes difficult even at rest, and especially after eating. When an individual becomes short of breath, his apprehension may make the air hunger even worse.

Some emphysematous patients complain of a chronic cough but this may be attributed to smoking, to environmental conditions or to recurrent airway infections which cause very thick sputum that is difficult to cough up (especially in the later stages of emphysema when the cough mechanism is ineffective). Other symptoms of emphysema may include fatigue, loss of weight, loss of appetite and loss of a sense of well-being. Patients generally describe a sense of fullness after meals and develop a dislike for high-caloric foods.

Hypoxemia is a reduction in oxygenation saturation of the arterial blood and is a condition often caused by pulmonary emphysema, but there is generally no constant relationship between the degree of hypoxemia and the degree of dyspnea experienced.(2,3) Inadequate pulmonary ventilation leads to an increase in the carbon dioxide content of arterial blood, a condition called hypercapnia which is

another characteristic of pulmonary emphysema. In severe diffuse pulmonary emphysema, the respiratory chemoreceptors eventually become insensitive to the hypercapnia condition, carbon dioxide stimulation of respiration is reduced, and the peripheral chemoreceptors respond to hypoxemia as a stimulant to respiration. Thus, care must be taken when administering oxygen therapy because if the patient's oxygen intake is increased too much, there will be no stimulation of respiration leading to further ventilatory failure.(12,13) Hypercapnia reduces blood pH, causing respiratory acidosis. With chronic acidosis, the emphysematous patient compensates with the blood buffer systems. However, when terminal emphysema results in overwhelming respiratory acidosis due to carbon dioxide intoxication, buffers are inadequate, resulting in death.

The capillary bed within the pulmonary circulation is reduced as a result of pulmonary tissue atrophy, with alveolar breakdown.(2,11) This leads to an increase in the resistance to blood flow, pulmonary hypertension, right heart strain and eventually cardiac failure. The prolonged and obstructed expiration that develops impedes venous return and right heart refilling; this also adds to the inefficiency of the heart.

The emphysematous patient's physical examination may reveal characteristic abnormalities such as the "barrel chest." (14) This is a condition described as an increased antero-posterior diameter which may equal or exceed the lateral diameter of the thoracic cage; the sternum is prominent; the thoracic spine becomes kyphotic; the ribs are elevated in the inspiratory position; and, there is very little

thoracic cage movement even during forced breathing. These conditions or characteristics are noted mostly during advanced pulmonary emphysema.

The combined use of x-rays, pulmonary function tests and arterial blood gas analysis will permit a fairly accurate diagnosis of emphysema and also provide a means of evaluating therapy.(15,16) Although chest x-rays alone cannot be used to definitely diagnose pulmonary emphysema, careful interpretation will indicate the areas of hyperinflation and the position of the diaphragm. Chest x-rays along with pulmonary function testing provides good support for the diagnosis of pulmonary emphysema.(7,13) The pulmonary function profile of pulmonary emphysema includes the loss of lung elastic recoil, the one factor which separates emphysema from the other COPDs; the ratio of residual volume (RV) to the total lung capacity (TLC) exceeds the normal value of 0.30; and the flow rates are decreased on forced expiration, thus promoting air-trapping and reduced ventilation.(3,17) The amount of oxygen and carbon dioxide dissolved in the arterial blood represents the performance of the pulmonary system, therefore arterial blood gas analysis is an important tool to determine the degree of oxygenation and ventilation.

Emphysema is an irreversible disease, but with proper therapy an emphysematous individual can lead a fairly normal life. A majority of the emphysematous patients die as a result of either a respiratory infection or cardiac failure; therefore, therapy must include measures which will produce symptomatic relief and reduce the potential for respiratory infection.(18,19) Pulmonary rehabilitation involves

breathing exercises where the patient's attention is directed towards abdominal breathing resulting in more diaphragmatic movement, thus increasing ventilation.(20) General exercise such as walking with and without oxygen is good for developing more efficient oxygen utilization and reduce the frequency of dyspnea.(21) Aerosol therapy and postural or gravity drainage will increase the liquification and the removal of the thick secretions, thus reducing the potential for obstruction and respiratory infection. Chest physical therapy may also include vibration and percussion (mechanical manipulations to assist in dislodging the secretions for evacuation).

Intermittent positive-pressure breathing (IPPB) devices are used to ventilate the lung with very little effort required by the patient. IPPB will benefit the emphysematous patient in three ways: (1) Hypoxia is relieved due to abundant air or oxygen forced into the lung; (2) the increased ventilation will reduce accumulated carbon dioxide; and (3) medications such as bronchodilators, mucolytics, detergents, wetting agents, etc. are nebulized and distributed throughout the respiratory system.(17,19) In advance cases of pulmonary emphysema where the patient has developed carbon dioxide narcosis due to severe hypercapnemia drastic measures must be taken. This includes endotracheal intubation or a tracheotomy if necessary; and, the patient is placed on a volume ventilator until the patient's blood gases are normalized and the manifestations of emphysema are brought under control.(17,22)

Chronic Bronchitis

Chronic bronchitis is defined as chronic excessive secretion of mucus in the tracheobronchial tree, which is not caused by specific diseases such as bronchiectasis or tuberculosis(2) and, chronic bronchitis is a disease of the peripheral airways where obstruction is caused not only by excess secretions but also by mucosal swelling and inflammation. It is often referred to as a syndrome rather than a disease because there are no adequate pathologic or physiologic criteria, therefore diagnosis is based primarily on the evidence of hypersecretion and chronic cough. It leads to many clinical features that are many times overlooked or misdiagnosed and as a result improper or insufficient treatment and follow-up is instituted.(9,10)

Chronic bronchitis develops over a period of years and is without symptoms in its earlier stages; therefore, the patient may be unaware of the onset of the disease. The patient's history generally consists of cough, sputum production, difficult expectoration especially in the morning, and, almost always, smoking.(23) In the later stages of chronic bronchitis inflammation and infections are more frequent and persistent resulting in the same manifestations and symptoms observed in emphysematous patients.

The patient's physical examination reveals a labored and prolonged inspiration and expiration with wheezing noises caused by bronchial narrowing.(5) Peripheral edema is noted in the patient's legs and ankles due to right ventricular failure which occurs much earlier in bronchitics than in emphysematous patients because airway

obstruction and increased pulmonary vascular resistance develops much faster in chronic bronchitis.(4)

Chest x-rays usually offer no reliable evidence for the diagnosis of chronic bronchitis; but, pulmonary function tests show decreased flowrates on inspiration and expiration, variable and normal RV/TLC ratios, and normal elastic recoil. Arterial blood gas analysis will indicate bouts of hypoxemia and hypercapnemia which occur for the same obstructive reasons previously discussed in emphysema.

Since the manifestations of chronic bronchitis are similar to those associated with emphysema, the therapy used to treat bronchitics is the same as that recommended for emphysematous patients. Emphasis is placed on the use of diuretics and low salt diets to reduce peripheral edema, and the use of prophylactic chemotherapy to prevent or control recurrent bacterial infections that usually occur during the winter months.(17,24) It is often difficult to treat a bronchitic because the physician requires complete cooperation of his patient in order to control environmental factors such as smoking; but, progress is slow and when the patient doesn't see dramatic results, he gives up and goes back to smoking.

Asthma

Asthma is defined as the presence of episodic, reversible airway obstruction produced by the following three mechanisms: bronchial smooth musculature contraction (bronchoconstriction), airway mucosal edema, and the presence of thick secretions in the airway.(3,25)

Asthmatic bronchitis is the most common type of asthma, but the factor common to all types of asthma is bronchoconstriction or bronchospasm.

Respiratory wheezing associated with asthma may also be heard in bronchogenic cancer, pulmonary embolism, tracheo-bronchial tuberculosis, or the presence of foreign bodies in the bronchi.(7)

Most of the causes and factors associated with asthma are unknown. A allergy, one of the known factors, has been studied quite extensively and is one of the few factors sufficiently tangible to support a basis for therapy, but even the intangibles of allergy are numerous. Bronchospasms or "asthmatic attacks" may be triggered by exposure to a specific allergen; but, the same individual may have attacks when angered, when inspiring cold air, following excessive exertion, or during respiratory tract infections.(25,26)

During normal ventilatory movement the bronchial lumen is widened during inspiration, narrowed during expiration and the exhaled air is removed passively due to the elastic recoil of the lung; but, when bronchospasm and mucosal edema occur, the lumen almost closes resulting in air trapping and labored expiration.

Expiratory wheezing is the most common clinical manifestation of asthma but during an asthma attack the manifestations may range from coughing spells and dyspnea on up to attacks so severe that oxygen deprivation with extreme cyanosis and perhaps unconsciousness could occur resulting in a major medical emergency called status asthmaticus.(26)

The physical examination may reveal nothing abnormal between asthmatic attacks; therefore, the physician may have to make the diagnosis on the basis of the patient's complaints.(7) In most asthmatics the physician can hear wheezing caused by mild

bronchospasms, so mild that the patient may be unaware of them. During an attack the patient will exhibit a prolonged and strenuous expiration; lower costal margin retractions during inspiration; and, because of hyperinflation, hyperresonant or tympanitic sounds during chest percussion.(17,19)

Chest x-rays give little support to the diagnosis of asthma because between asthma attacks the chest may appear normal. However, if the chest film is obtained during a severe attack it may look similar to an advanced state of pulmonary emphysema; that is, a flat diaphragm and ribs which have elevated and separated. This similarity has led to incorrect diagnosis; therefore, it is important to obtain x-rays during an attack and when there are no asthmatic symptoms.(25,26) The pulmonary function profile shows that most asthmatics have normal elastic recoil, increased RV/TLC ratios, and decreased inspiratory and expiratory flowrates which, unlike altered flowrates seen in emphysema or chronic bronchitis, can be reversed with bronchodilator medication. Thus, a pulmonary function study before and after bronchodilators is a key diagnostic aid for asthma. (7,15) Arterial blood gas analysis will not show anything significant unless the asthmatic attack is severe causing impaired ventilation to the point where oxygenation is reduced and carbon dioxide is retained.

The treatment of asthma is generally aimed at the symptomatic relief of the acute asthmatic attack with subsequent treatments designed to prevent attack recurrence.(17) The acute attack is treated by the administration of bronchodilators by either injection

or inhalation (aerosol) depending on the patient and the severity of the attack. If the patient cannot breath deep enough voluntarily for good aerosol deposition, IPPB may be necessary to force the aerosol deep into the lungs. Bronchodilators such as epinephrine and isuprel are designed to relax the smooth musculature of the respiratory airways thus reducing bronchospasms. Caution must be taken when administering bronchodilators to patients with questionable circulatory disease because these drugs can produce sympathomimetic effects such as tachycardia, excitement, tremors and dizziness.(7,17) Steroids or cortisone drugs are used to reduce airway inflammation; however, prolonged use of steroids could result in obesity, electrolyte imbalance, edema, acne and osteoporosis.(25,26) A low percentage of oxygen (24-28%) may be given via nasal cannula or Venturi (oxygen dilutor) mask to treat arterial hypoxemia.(17,27)

Small Airways Disease

Small airways disease involves the inflammation of the very small airways such as the bronchioles - airways less than two millimeters in diameter.(3) It is not known whether this inflammation is reversible in all patients, whether it is an early phase of chronic bronchitis or emphysema; or just where it fits into the whole COPD picture. For this reason I wish to leave this category out of my study.

Air Pollution

Through various screening programs and studies of large cities, the incidence of death caused by complications of chronic bronchitis and emphysema due to air pollution has been estimated. In New York

between 1961 and 1971 the death rate from emphysema had increased 500 percent while deaths from chronic bronchitis increased 200 percent; and, before 1965 nearly 15,000 individuals were receiving disability compensation because of the complications of emphysema.(28)

There are many more statistics available and they all lead to the conclusion that the increase in the incidence of chronic bronchitis and emphysema is the result of the rising concentration of pollutants and irritants in the air. The causes of air pollution are generally classed as environmental, occupational, and personal. Automobile exhaust and industrial combustion are examples of environmental pollution. Industrial fumes and mill waste products such as dust are examples of occupational pollution. Tobacco products such as cigarettes are examples of personal pollutants.

The effects of tobacco consumption have been summarized in "The Health Consequences of Smoking, a report of the Surgeon General: 1971." Some of the more important conclusions are listed below.(23)

1. The risk of dying from chronic bronchitis increases with cigarette smoking.
2. An increased risk of dying from emphysema is associated with cigarette smoking.
3. In the United States, the importance of cigarette smoking as a cause of COPD is much greater than that of atmospheric or occupational pollution.
4. Cigarette smokers suffer from cough, sputum production or the combination of the two more frequent than non-smokers.

5. A reduction in ventilatory function is associated with cigarette smoking.
6. Male cigarette smokers have a greater prevalence of dyspnea than non-smokers.
7. A lower mortality risk follows those who stop cigarette smoking relative to those who continue to smoke.
8. Pipe and cigar smokers are generally much less affected than cigarette smokers by COPD.
9. Respiratory symptoms and reduced ventilatory function has been demonstrated frequently even in relatively young cigarette smokers.

The increasing number of people with chronic bronchitis and emphysema is actually the product of our civilization. The luxuries and benefits of industrialization have been accompanied by the hazards of both environmental and occupational pollution. Our bountiful society and its status symbols have made cars a way of life and cigarette smoking the socially accepted method of reducing the tensions created by such a society.

Purpose

Disease descriptions can be very detailed and verbose, but this author intended only to give the reader a brief idea about what is involved with COPD and its associated follow-up. The chronic obstructive pulmonary diseases have managed to escape any real advances in diagnosis and proper management. The knowledge generated at research hospitals and large medical centers seems to remain at these places

and most of the studies appear to be concentrated in the area of the critically ill COPD patient. However, most of the COPD patients are not that critical and are admitted to the smaller, less research-oriented hospitals creating the question that has stimulated this research paper: What happens to these patients?

There are a number of general and specific objectives; the general objectives include:

1. The development of a method for auditing COPD medical records.
2. The determination of areas for future investigation and research.

The specific objectives center around an exploratory analysis and correlation study which will be utilized to determine the progress on admitting procedures and follow-up care for those individuals afflicted with COPD.

It is the intention of this author to establish some trends in the care of these patients; to determine if the care has improved over a three year period; to identify those areas of therapy which could be improved; and to correlate various vital and personal information associated with the COPD patient. These results will be compared to those guidelines for the treatment of pulmonary diseases established by the Joint Committee on Professional Standards Review (PSRO). It must be remembered that these guidelines vary depending on the patient, the doctor and the services and facilities that are available.

MATERIALS AND METHODS

The primary source of data for this study was obtained from the patient's medical record. The components of a medical record in approximate order include: a personal statistics or identification sheet, a clinical resume, or summary of medical care, an emergency room record if the patient was admitted there, a readmission note if the patient was previously hospitalized, the patient's history and physical examination report, progress notes or day to day comments stated by the physician about the patient, physician's order or prescription forms, laboratory test report forms, posting slips stating the results of treatment and therapy, nurses' notes, and miscellaneous charts and insurance forms. The data sheet shown in Figure 1 was developed to permit extraction of information from medical records on one page, making keypunching of data easier.

Data were extracted from medical records of 202 former patients of Orange Memorial Hospital in Orlando, Florida. Records were selected so as to provide an equal number of the disease categories dispersed equally over the two years. One hundred records were dated 1973 and 102 were dated 1976. Emphysema, chronic bronchitis, and asthma had been diagnosed in 67, 67, and 68 of the cases respectively. These two years were chosen because they were before and after the period (1974-1975) when some of the greatest advances in pulmonary care were made including oxygenation and ventilator techniques. Medical records

FIGURE 1

Data Sheet for Medical Record Auditing

1. Case # _____	
2. Diagnosis: 1. Emphysema 2. Asthma 3. Chronic Bronchitis 4. Small Airway Disease - - -2.	<input type="checkbox"/>
3. Sex: 1. Male 2. Female - - - - -3.	<input type="checkbox"/>
4. Age: (years) 1. <20 2. 21-30 3. 31-40 4. 41-50 5. 51-60 6. 61-70 7. >70 - - - - -4.	<input type="checkbox"/>
5. Length of stay: 1. <5 2. 5-10 3. 11-15 (days) 4. 16-20 5. 21-25 6. >25 - - - - -5.	<input type="checkbox"/>
6. Chief complaint: 1. Short of breath 2. Edema of Appendages 3. 1 & 2 - - - - -6.	<input type="checkbox"/>
7. Type of work (greatest % of time) 1. Desk job 2. Factory 3. Farming 4. Construction 5. None - - -7.	<input type="checkbox"/>
8. Smoker: 1. Yes 2. No - - - - -8.	<input type="checkbox"/>
8a. If yes, how long: (years) 1. <10 2. 10-15 3. 16-20 4. 21-25 5. 26-30 6. >30 - - - - -8a.	<input type="checkbox"/>
8b. If yes, what type: 1. Cigarette 2. Cigar 3. Pipe - - - - -8b.	<input type="checkbox"/>
9. Heart Trouble: 1. Infarctions 2. Right Heart Failure 3. Left Heart Failure 4. None - - - - -9.	<input type="checkbox"/>
10. Mechanical Ventilation: 1. IPPB 2. Long Term 3. 1 & 2 4. None - - - - -10.	<input type="checkbox"/>
10a. If IPPB: 1. BID 2. TID 3. QID 4. >QID - - - - -10a.	<input type="checkbox"/>
10b. If long term, type of airway: 1. Trach 2. Nasal Ent 3. Oral Ent 4. Mask - - - - -10b.	<input type="checkbox"/>
10c. Length of time: (days) 1. <1 2. 1-5 3. 6-10 4. 10-15 5. >15 - - - - -10c.	<input type="checkbox"/>
10d. Ventilator Modifications 1. PEEP 2. IMV 3. 1 & 2-10d.	<input type="checkbox"/>
11. Respiratory Drugs. 1. Bronchodilators 2. Mucolytics 3. Antibiotics 4. Steroids 5. Digitalis 6. 1 & 2 7. 1 & 3 8. 1 & 4 9. 2 & 3 10. 2 & 4 11. 3 & 4 -11.	<input type="checkbox"/>
12. Pulmonary Rehabilitation: 1. Yes 2. No - - - - -12.	<input type="checkbox"/>
Test done on admission:	
13a. Pulmonary functions 1. Yes 2. No - - - - -13a.	<input type="checkbox"/>
13b. Chest films 1. Yes 2. No - - - - -13b.	<input type="checkbox"/>
13c. Arterial Blood gases 1. Yes 2. No - - - - -13c.	<input type="checkbox"/>

were chosen arbitrarily from each of the previously mentioned disease categories by a records custodian. The total number of patients admitted diagnosed as having emphysema, chronic bronchitis and asthma was 130, 48 and 93 in 1973, and 78, 34 and 99 in 1976 respectively.

The data was keypunched, one card for each data sheet, and run on a computer using the Statistical Package for the Social Sciences as the program.(29) Frequency distributions of patients were run in all categories shown on data sheet (Figure 1) and all data was crosstabulated by diagnosis, smoking and year. The chi square test was applied to all crosstabulations, and that data not within the 95% confidence limit was excluded from the project. The remaining results were analyzed to evaluate the relative effectiveness of the various treatment profiles prescribed for patients in various aspects of their admission, care and therapy. Working with medical record data of this type resulted in limited use of statistics, therefore absolute count and percentages were primarily used resulting in a descriptive type research paper.

RESULTS AND DISCUSSION

Frequency Distributions

The number of patients arranged according to year, diagnosis, sex and race are given in Table 1. One hundred patients from 1973 and 102 from 1976 were selected so that 67 patients were diagnosed as emphysematous, 68 were diagnosed as asthmatic and 67 were diagnosed as chronic bronchitics. These data were expected because of the sampling method used in this study and no other criteria were selected for.

There were 109 males (54%) and 93 females (46%) in this study. This proportion is typical as indicated by various reports and studies done on smoking and chronic obstructive lung diseases. (23,30) The published percentage fluctuates tremendously but there are usually more males than females associated with COPD possibly because there are more male smokers than female smokers. (23)

There were 172 whites (85%) and 30 non-whites (15%) in this study. The one possible explanation for this ratio may be due to those physicians with admitting privileges at Orange Memorial Hospital, probably having more whites than non-whites as clients.

The number of patients arranged by the length of their hospital stay, their complaints on admission and smoking habits are given in Table 2. It appears that the greatest percentage of patients (40%) were hospitalized at Orange Memorial between 5 and 10 days, and the next greatest percentage were hospitalized less than 5 days. The primary patient complaint appears to be shortness of breath (76%).

TABLE 1

Frequency distributions of patients
for year, diagnosis, sex and race

Category	Absolute Frequency (#)	Relative Frequency (PCT)
<hr/>		
Year		
1973	100	49.5
1976	<u>102</u>	<u>50.5</u>
Total	202	100.0
<hr/>		
Diagnosis		
Emphysema	67	33.2
Asthma	68	33.7
Bronchitis	<u>67</u>	<u>33.2</u>
Total	202	100.0
<hr/>		
Sex		
Male	109	54.0
Female	<u>93</u>	<u>46.0</u>
Total	202	100.0
<hr/>		
Race		
White	172	85.1
Non-white	<u>30</u>	<u>14.9</u>
Total	202	100.0

TABLE 2

Frequency distributions of patients
for hospital stay, patient complaints and smoking habits

Category	Absolute Frequency (#)	Relative Frequency (PCT)
<hr/>		
Length of stay (days)		
<5	78	38.6
5-10	80	39.6
11-15	24	11.9
16-20	9	4.5
21-25	7	3.5
>25	<u>4</u>	<u>2.0</u>
Total	202	100.0
<hr/>		
Patient complaints		
Short of breath (only)	154	76.2
Edema of limbs (only)	0	0
Both of the above	10	5.0
Not documented	<u>38</u>	<u>18.8</u>
Total	202	100.0
<hr/>		
Smoking habits (past or present)		
Yes	99	49.0
No	65	32.2
Not indicated	<u>38</u>	<u>18.8</u>
Total	202	100.0
<hr/>		

Being short of breath frightens people and they in turn come to the emergency room or the doctor's office for relief. Many of these patients may have edema of the legs and ankles but the shortness of breath may cause them to be unaware of it. Once the individual is under medical supervision, his or her recovery is very quick; hospital control of the patient's living habits, diet and activity along with the prescribed medications, apparently keeps the majority of the hospital visits under 10 days.

At least forty-nine percent of those patients with COPD had or presently have smoking habits. It's amazing that the records did not indicate whether the patient was a smoker in 19% of the patients. Personal observation of patients in a hospital or at home under the doctor's care for COPD indicates that a surprising number still smoke, even after severe bouts of respiratory distress.

The number of patients given mechanical ventilation, intermittent positive pressure breathing (IPPB) and some type of airway are given in Table 3. The majority of the patients that required mechanical ventilation had IPPB (56%). These patients apparently required only short term assisted ventilation to relieve them of their distress and to administer perscribed aerosolized medications. A large portion (40%) of the patients admitted required no mechanical ventilator, but may require some nebulization therapy and/or supplemental oxygen therapy. Antibiotic treatment may have been all that was required in some cases. Only a very small percentage (4.5%) of patients required some long term ventilation, perhaps because of properly instituted preventative medical care.

TABLE 3

Frequency distributions of patients arranged by
type of mechanical ventilation, frequency of IPPB and type of airway

Category	Absolute Frequency (#)	Relative Frequency (PCT)
<hr/>		
Mechanical ventilation		
IPPB	113	55.9
Long term	1	0.5
Both	8	4.0
None	<u>80</u>	<u>39.6</u>
Total	202	100.0
<hr/>		
IPPB daily frequency		
2 times/day	12	5.9
3 times/day	9	4.5
4 times/day	84	41.6
> 4 times/day	16	7.9
No IPPB	<u>81</u>	<u>40.1</u>
Total	202	100.0
<hr/>		
Type of Airway		
Trach tube	7	3.5
Nasal endotracheal	0	0
Oral endotracheal	5	2.5
No airway	<u>190</u>	<u>94.0</u>
Total	202	100.0
<hr/>		

Almost 42% of the patients on short term IPPB therapy received their treatments 4 times a day (QID). The frequency of IPPB is generally determined by the nature of the medications used in the IPPB machine. Bronchodilators have a duration of action between 3 and 4 hours. Patients not receiving any special type of medications such as bronchodilators may require IPPB only 2 (BID) or 3 (TID) times a day, thus serving primarily as a hyperventilator and oxygen delivery device. Thus, it appears that most were on bronchodilators.

Twelve patients (6%) required some type of modified airway either a tracheotomy or an oral endotracheal tube. Unless the patient is going to be on supportive ventilation for longer than 3 or 4 days, oral tubes are usually sufficient if properly monitored and maintained. (17) There were no nasal endotracheal tubes used in this study probably because it is much easier and quicker to place the oral tube especially during emergency situations.

Crosstabulations of Diagnosis and of Smoker

Emphysema, asthma and bronchitis were crosstabulated by sex and by age resulting in data given in Table 4. When reading tables of this type one would characterize the components of the diagnosis category by reading the percentages across from row percent, and one would characterize the components in the horizontal direction by reading the percentages vertically in the column percent. For example, the emphysematous patients in this study were primarily male (75%), the asthmatics were mostly female (68%). Of all the males in this study 46% had emphysema and of all the females 50% had asthma. The dominant

TABLE 4

Crosstabulations of diagnosis by sex and by age

Diagnosis	Sex	
	Male	Female
Emphysema	50	17
Row (PCT)	74.6	25.4
Column (PCT)	46.3	17.4
Asthma	22	46
Row (PCT)	32.4	67.6
Column (PCT)	20.4	50.0
Bronchitis	36	30
Row (PCT)	55.7	44.3
Column (PCT)	33.3	32.6

Diagnosis	Age (Years)						
	<20	20-30	31-40	41-50	51-60	61-70	>70
Emphysema	0	1	0	5	25	17	19
Row (PCT)	0.0	1.5	0.0	7.5	37.3	25.4	28.4
Column (PCT)	0.0	5.0	0.0	20.8	48.1	50.0	52.8
Asthma	20	12	9	6	12	4	5
Row (PCT)	29.4	17.6	13.2	8.8	17.6	5.9	7.4
Column (PCT)	95.2	60.0	60.0	25.0	23.1	11.8	13.9
Bronchitis	1	7	6	13	15	13	12
Row (PCT)	1.5	10.4	9.0	19.4	22.4	19.4	33.3
Column (PCT)	4.8	35.5	40.0	54.2	28.8	38.2	33.3

age group for emphysema appears to be between 50 and 60 years (37%) and emphysema seems to be the major diagnosis (48% - 53%) in those patients older than 50 years. Almost one-third (29%) of the asthmatics were less than 20 years old perhaps because asthma is a type of COPD that occurs at a younger age and is diagnosed early. (25,26) Bronchitis appears to be more dominant between the ages of 40 and 50 years (54%). Since bronchitis is similar to emphysema in progressive characteristics it makes sense that the age distributions were also similar. (31)

Crosstabulations of diagnosis by length of hospital stay and by occupation are given in Table 5, and crosstabulations of smoker by occupation are given in Table 6. It appears that most of the emphysematous patients, asthmatics, and bronchitics were hospitalized less than 15 days, 10 days and 10 days respectively; and, it seems that those patients having extended hospital stays, longer than 20 days, were diagnosed primarily as emphysematous (56% - 100%). Fifty percent of the asthmatics were hospitalized less than 5 days and many of these patients' records indicated a stay of usually 1 or 2 days, perhaps due to the fairly rapid relief of the bronchospasms. Forty-six percent of the bronchitics stay in the hospital between 5 and 10 days. The longer hospitalization associated with emphysema and bronchitis was probably due to hypoxia, prolonged increased work of breathing and severe obstruction due to secretions.

Most of the emphysematous patients that indicated some type of occupation worked in an office (41%). There were 31% in this disease category that indicated no occupation but were designated on the

TABLE 5

Crosstabulations of diagnosis by
length of hospital stay and by occupation

Length of Stay (Days)						
Diagnosis	<5	5-10	11-15	16-20	21-25	>25
Emphysema	19	18	16	5	5	4
Row (PCT)	28.4	26.9	23.9	7.5	7.5	6.0
Column (PCT)	24.4	22.5	66.7	55.6	71.4	100.0
Asthma	34	31	2	0	1	0
Row (PCT)	50.0	45.6	2.9	0.0	1.5	0.0
Column (PCT)	43.6	38.8	8.3	0.0	14.3	0.0
Bronchitis	25	31	6	4	1	0
Row (PCT)	37.3	46.3	9.0	6.0	1.5	0.0
Column (PCT)	32.1	38.8	25.0	44.4	14.3	0.0

Occupation						
Diagnosis	Desk	Factory	Farming	Construction	None	
Emphysems	17	4	2	6	13	
Row (PCT)	40.5	9.5	4.8	14.3	31.0	
Column (PCT)	37.8	25.0	50.0	42.9	18.6	
Asthma	12	4	1	2	37	
Row (PCT)	21.4	7.1	1.8	3.6	66.1	
Column (PCT)	26.7	25.0	25.0	14.3	52.9	
Bronchitis	16	8	1	6	20	
Row (PCT)	31.4	15.7	2.0	11.8	39.2	
Column (PCT)	35.6	50.0	25.0	42.9	28.6	

medical record as being retired or housewives. Of those patients who were office workers 38% had emphysema but the highest frequency occurred in the farming category (50%) which is misleading because there only 4 farmers in this study. The majority of the asthmatics (66%) indicated no occupation, perhaps because the age distribution less than 20 years was so high (Table 4), leading one to believe that asthma is not occupationally related. Of those asthmatics that did indicate some occupation, 21% were office employees which seemed feasible since most of the asthmatics were female (Table 4). A large percentage (31%) of bronchitics were found to be office employees, and 39% indicated no occupation. It was observed that many of the bronchitics like the emphysematous patients that indicated no occupation were designated on the medical record as being retired or housewives. Construction workers were evenly distributed between bronchitics and emphysematous patients, both categories indicating 43%. The majority of the factory workers (50% were bronchitics and the majority of the farmers (50%) were emphysematous, probably due to the two things that all three occupations have in common, dust and irritating inhalants.(28)

Another possible occupational hazard appears to be smoking (Table 6). The majority of the patients that smoked were desk or office employees (44%) which could be correlated with the high frequency of emphysema and bronchitis associated with office employment (Table 5). Although the highest frequency of smokers were office employees, no less than 71% of the patients in the other occupational categories in this study were also smokers.

TABLE 6
Crosstabulations of Smoker by Occupation

Smoker [*]	Occupation				
	Desk	Factory	Farming	Construction	None
Yes	31	10	4	10	15
Row (PCT)	44.3	14.3	5.7	14.3	21.4
Column (PCT)	83.8	71.4	100.0	90.0	26.3
No	6	4	0	1	42
Row (PCT)	11.3	7.5	0.0	1.9	79.2
Column (PCT)	16.2	28.6	0.0	0.8	43.1

* There were 79 missing observations resulting in some Column (PCT) not totalling 100%.

TABLE 7

Crosstabulations of diagnosis by
smoking habits and by length of time smoking

Smoking Habits						
Diagnosis	Yes	No				
Emphysema	52	2				
Row (PCT)	94.5	3.6				
Column (PCT)	53.1	3.1				
Asthma	13	47				
Row (PCT)	21.7	78.3				
Column (PCT)	13.3	72.3				
Bronchitis	33	16				
Row (PCT)	67.3	32.7				
Column (PCT)	33.7	24.6				

Length of time smoking (years)						
Diagnosis	<10	10-15	16-20	21-25	26-30	>30
Emphysema	1	0	1	3	14	26
Row (PCT)	2.2	0.0	2.2	6.7	31.1	57.8
Column (PCT)	25.0	0.0	20.0	42.9	73.7	63.4
Asthma	2	2	1	2	2	1
Row (PCT)	20.0	20.0	10.0	20.0	20.0	10.0
Column (PCT)	50.0	40.0	20.0	28.0	10.5	2.4
Bronchitis	1	3	3	2	3	14
Row (PCT)	3.8	11.5	11.5	7.7	11.5	53.8
Column (PCT)	25.0	60.0	60.0	28.6	15.8	34.1

Crosstabulations of diagnosis by smoking habits and by length of time smoking are given in Table 7. As already indicated in Table 2, the frequency of smoking habits was high (49%) in those patients who were questioned about their social history. In Table 7 it appears that most of the emphysematous patients smoked (95%), almost 90% smoked longer than 25 years, and 58% smoked over 30 years. The majority of the patients smoking longer than 25 years had emphysema (74%) resulting in a fairly strong correlation between emphysema and smoking as a causative factor. Seventy eight percent of the asthmatics were non-smokers with 72% of all the non-smokers being asthmatics. The tendency for asthmatics not to smoke is probably because of the respiratory difficulty they have earlier in their lives.(25) Those asthmatics who indicated smoking habits appeared to have a fairly even distribution throughout the time span. Bronchitics, like the emphysematous patients, had a high frequency (67%) of smoking habits and note that the greatest percentage (60%) of the patients smoking between 10 and 20 years had bronchitis. This may be attributed to the fact that respiratory symptoms show up sooner in bronchitics than in emphysematous patients.(3,4) Like emphysematous patients the majority (54%) of the bronchitics that indicated smoking habits smoked longer than 30 years which could also result in a fairly strong correlation between bronchitis and smoking as a causative factor.

Crosstabulations of diagnosis by mechanical ventilation and by pulmonary rehabilitation are given in Table 8. Patients with emphysema had a high frequency of IPPB (75%) with an additional 13% requiring long term mechanical ventilation with IPPB. The asthmatics in this

TABLE 8

Crosstabulations of diagnosis by
mechanical ventilation and by pulmonary rehabilitation

Mechanical Ventilation				
Diagnosis	IPPB	Long Term	Both	None
Emphsema	38	1	8	20
Row (PCT)	56.7	1.5	11.9	29.9
Column (PCT)	33.6	100.0	100.0	25.6
Asthma	49	0	0	17
Row (PCT)	74.2	0.0	0.0	25.8
Column (PCT)	43.4	0.0	0.0	21.8
Bronchitis	36	0	0	41
Row (PCT)	38.8	0.0	0.0	61.2
Column (PCT)	23.0	0.0	0.0	52.6

Pulmonary Rehabilitation				
Diagnosis	Yes	No		
Emphysema	21	46		
Row (PCT)	31.3	68.7		
Column (PCT)	60.0	27.5		
Asthma	5	63		
Row (PCT)	7.4	92.6		
Column (PCT)	14.3	37.7		
Bronchitis	9	58		
Row (PCT)	13.4	86.6		
Column (PCT)	25.7	34.7		

study required no long term ventilation but had a high frequency of IPPB, the asthmatics had the highest frequency (43%) with emphysema being second (34%). The bronchitics also required no long term ventilation but had some IPPB (39%). The low frequency of long term ventilation in all three categories was perhaps due to properly instituted medical care which kept a majority of the patients' conditions from becoming worse.

The utilization of pulmonary rehabilitation; that is, breathing exercises and chest physio therapy in emphysema, asthma and bronchitis was relatively low with frequencies of 31%, 7% and 13% respectively; but, of all the patients who received pulmonary rehabilitation, 60% were emphysematous. Because of the secretion retention problems associated with emphysema and bronchitis, one would think that the frequency of pulmonary rehabilitation should be higher.(3,4)

Crosstabulations of diagnosis by race and by bronchospasms are given in Table 9. Most of the emphysematous patients were white (94%), 37% of which had emphysema, and very few of these patients (2%) had bronchospasms. The greatest percentage of asthmatics were also white (74%) with a high frequency of bronchospasms (68%) and the majority of those patients who had bronchospasms were asthmatics (96%). Like the other two disease categories, the bronchitics were primarily white (88% and approximately 1/3 of the white patients (34%) had bronchitis. The frequency of bronchospasms associated with bronchitis was the same as that observed in the emphysematous patients (2%). As stated earlier in the sections on frequency distributions (Table 1) the possible explanation for the high frequency of white patients

TABLE 9

Crosstabulations of diagnosis by
race and by bronchospasms

Diagnosis	White	Non-White
Emphysema	63	4
Row (PCT)	94.0	6.0
Column (PCT)	36.6	13.3
Asthma	50	18
Row (PCT)	78.5	26.5
Column (PCT)	29.1	60.0
Bronchitis	59	8
Row (PCT)	88.1	11.9
Column (PCT)	34.3	26.7

Bronchospasms		
Diagnosis	Yes	No
Emphysema	1	66
Row (PCT)	1.5	98.5
Column (PCT)	2.1	42.9
Asthma	46	22
Row (PCT)	67.6	32.4
Column (PCT)	95.8	14.3
Bronchitis	1	66
Row (PCT)	1.5	98.5
Column (PCT)	2.1	42.9

TABLE 10

Crosstabulations of diagnosis by death

Diagnosis	Death	
	Yes	No
Emphysema	11	56
Row (PCT)	16.4	83.6
Column (PCT)	91.7	29.5
Asthma	0	68
Row (PCT)	0.0	100.0
Column (PCT)	0.0	35.8
Bronchitis	1	66
Row (PCT)	1.5	98.5
Column (PCT)	8.3	34.7

associated with these three diseases is perhaps those physicians with admitting privileges at Orange Memorial Hospital, probably have more white than non-white clients. The high percentages of pronchospasms observed in the asthmatics would be expected. (25,26)

Crosstabulations of diagnosis by death are given in Table 10. There were no deaths associated with asthma in this study and only 2% of the bronchitics died. Ninety-two percent of the patients that died had emphysema, yet of those patients suffering from emphysema 16% died.

Crosstabulations of year

Crosstabulations of year from which the data was collected by time on ventilator and by ventilator modifications are given in Table 11. In 1973, 80% of the patients requiring long term ventilation spent 10 to 15 days on the ventilator; and, in 1976 almost 57% of the patients on long term ventilation spent less than 5 days on the ventilator. There were 75% - 100% of the patients spending less than 5 days on the ventilator in 1976. It is now realized that it is important to get the patient off the ventilator as soon as possible because the patient can become psychologically and physiologically dependent on the ventilator. (17) In 1976, as compared to 1973, it appears that those patients capable of being weaned from the ventilator were done so sooner. Intermittant mandatory ventilation (IMV) is characterized by the patient breathing spontaneously alternated by positive pressure mechanical ventilation placing a majority of the ventilatory workload on the patient, thus creating a type of weaning method. Positive end expiratory pressure (PEEP) is used to produce back pressure in the respiratory system to facilitate gas exchange.

TABLE 11

Crosstabulations of year by
time on ventilator and by ventilator modifications

Time on ventilator (days)					
Year	<1	1-5	5-10	10-15	>15
1973	0	1	0	4	0
Row (PCT)	0.0	20.0	0.0	80.0	0.0
Column (PCT)	0.0	25.0	0.0	100.0	0.0
1976	1	3	0	0	3
Row (PCT)	14.3	42.9	0.0	0.0	42.9
Column (PCT)	100.0	75.0	0.0	0.0	100.0
Ventilator modifications					
Year	Positive end expiratory pressure (PEEP)	Intermittant mandatory ventilation (IMV)		Both	
1973	0	0		1	
Row (PCT)	0.0	0.0		100.0	
Column (PCT)	0.0	0.0		100.0	
1976	1	6		0	
Row (PCT)	14.3	85.7		0.0	
Column (PCT)	100.0	100.0		0.0	

TABLE 12

Crosstabulations of year by blood
gases and by controlled oxygen administration

Blood gases		
Year	Yes	No
1973	38	61
Row (PCT)	38.4	61.6
Column (PCT)	35.2	65.6
1976	70	32
Row (PCT)	68.6	31.4
Column (PCT)	64.8	34.4

Oxygen administration		
Year	Controlled	Uncontrolled
1973	15	30
Row (PCT)	33.3	66.7
Column (PCT)	24.6	81.1
1976	46	7
Row (PCT)	86.8	13.2
Column (PCT)	75.4	18.9

IMV and PEEP were prescribed for only one patient in this study in 1973, perhaps because it has been my observation that these two modifications only started to appear clinically at Orange Memorial Hospital around 1973. In 1976, IMV was 86% of the ventilator modification; and, even though there were very few patients involved between the two year a trend towards more utilization of these methods was noted.

Crosstabulations of year by blood gases and by controlled oxygen administration are given in Table 12. Sixty-five percent of the blood gas studies performed were done in 1976 as compared to 35% done in 1973. This may be due to the fact that there was a recommendation in 1974 from the medical committee at Orange Memorial Hospital which stated that all COPD patients admitted to this hospital must have an arterial blood gas study performed. The increase in blood gas studies could lead one to believe that the COPD patients are being monitored better now than in 1973 and this could attribute to the shorter time required on the ventilator (Table 11). Controlled oxygen administration implies that only enough oxygen is utilized to satisfy the hypoxic needs of the patient, and uncontrolled implies the administration of relatively inappropriate amounts of oxygen. In 1973, 67% of the patients receiving oxygen were given uncontrolled amounts and in 1976, 87% were receiving controlled amounts of oxygen either by a venturi mask or a low flow cannula. It appears that over the three-year span, the COPD patients were treated with oxygen more conservatively presumably because hypoxic phenomena (12,13) is more understood by those caring for the patient.

In summary, the typical emphysematous patient and bronchitic will

most likely be a white male over 50 years old and will probably be some type of office employee with smoking habits for over 25 years. He may complain of shortness of breath; and, if hospitalized, will spend 10 days or less treated four times a day with IPPB, little or no pulmonary rehabilitation, and a good chance of surviving. The typical asthmatic will most likely be a white female younger than 21 years old and will probably have no occupation and no smoking habits. He or she may complain of bronchospasms and shortness of breath; and, if hospitalized, will spend 10 days or less treated four times a day with IPPB, little or no pulmonary rehabilitation, and a good chance of surviving.

From 1973 to 1976, there was an increase in the frequency of arterial blood gas studies, PEEP, IMV, and controlled oxygen administration; and, there was a decrease in the amount of time the COPD patient spend on the ventilator.

Conclusion

There were many problems and variables associated with medical record sampling in this study. Particular care was taken to insure that the same patient was not evaluated more than once to insure a good sampling population. One must realize that each time an individual is admitted to the hospital, he or she receives a new case number resulting in 2 or 3 case numbers being the same patient, therefore a name check must be done. There were many incomplete records because there were patient history and physical examination forms only partially filled out and some were completely blank which lessened the effectiveness of this study. If these forms were completed, much personal and medical data could have been obtained which would reduce the number of missing observations. The physicians usually know their patient's history and physical status and this is perhaps why documentation is limited in this section of the medical record; but, it is obvious that it makes auditing of medical records difficult and quite limited. Poor medical record documentation does not imply poor patient care.

The data sheet used made sampling and keypunch preparation easier and much of the data which includes laboratory tests and diagnostic procedures was validated with the aid of report slips posted in the patient record. Medical record techniques and organization appeared to improve between 1973 and 1976 because patient information was easier to obtain from the records dated 1976 as compared to those dated 1973.

The majority of the results obtained from the various crosstabulations was for the most part expected except that there was a fairly low frequency of pulmonary rehabilitation which is one procedure that should be performed more. The patterns and characteristics of those patients suffering from chronic obstructive pulmonary disease were comparable to those patterns and characteristics stated in the cited literature.

The physicians at Orange Memorial Hospital appeared to vary in their understanding of the manifestations associated with chronic obstructive pulmonary disease. This appeared more obvious in 1973; but, perhaps due to better coordination between the respiratory therapists and the physicians, the care of the patient improved over the three-year period. Successful patient care seems to be a team effort.

More medical record audits should be performed and an additional study such as this one should be done with a longer time span, perhaps 6 or 8 years rather than 3 years. Much can be learned about the type of care that the patient is receiving and the type of improvements that could be made in medical record administration.

Based on this study, it is the opinion of this author that medical attention and documentation is headed in the proper direction.

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